LANDRY'S PARALYSIS.*

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In a paper presented to this Association two years ago, one of us (Thomas) from a study of the anatomical changes in two cases of acute ascending paralysis and of the cases with autopsies reported in the literature of the subject, came to the conclusion that the anatomical basis of the disease was an acute parenchymatous degeneration involving the peripheral motor neurone, arising from some toxic or infectious cause. In a paper presented to the Association a year ago, Mills and Spiller[2] came independently to a similar conclusion. The results of the autopsy in a third case which we present to-day are confirmatory of the conclusion previously reached.

As the evidence in favor of this conclusion is now so strong it seems hardly necessary to review in detail the results of a considerable number of autopsies reported since these two papers were published. Krewer[3] in three cases found subacute neuritis with acute myelitis; Schultz,[4] in one case acute diffuse myelitis with chronic neuritis; Roger and Josué[5] found changes in the anterior horns and pneumococci; Piccinino,[6] changes in the gray matter of the cord with the diplococcus intracellularis; Worcester,[7] changes in the anterior horns, peripheral nerves and cerebral cortex; Hirtz and Lesné,[8] changes in the anterior horns without any neuritis; Burghart,[9] in a case that had nearly recovered from the paralysis, changes in the anterior horns with very slight changes in the endoneurium; and Chantemesse and Ramond,[10] in two cases which perhaps should not be classed as Landry's paralysis, but as beri-beri, changes in both cord and nerves, with bacilli in the cerebrospinal fluid which produced similar symptoms in animals. In favor of the toxic—infectious origin, moreover, beside the evidence of the clinical cases, Remlinger[11] claims to have produced acute ascending paralysis in rabbits by inoculation with pathogenic cocci, and Rendu[12] has observed the symptoms developing after inoculation of anti-rabic serum.

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[1] Thomas, Amer. Journ. of the Med. Sciences, August, 1898.

[2] Mills and Spiller, JOURN. OF NERVOUS AND MENTAL DISEASE, June, 1898.

[3] Krewer, Zeitschrift für klin. Med., xxxii, 115.

[4] Schultz, Norsk Mag. f. Lagevid, No. 6, 1898, abstract in Revue Neurologique, 15 Jan., 1899.

[5] Roger and Josué, Press méd., 27 July, 1898.

[6] Piccinino, Annali di Neurologia, xv, 1, 1897.

[7] Worcester, JOURNAL of NERVOUS AND MENTAL DISEASE, May, 1898.

[8] Hirtz and Lesné, Presse méd., 12 June, 1897.

Although the affection is probably of toxic-infectious origin, the evidence in favor of a bacterial origin, in spite of Remlinger's experiments and the cases reported by Roger and Josué, Chantemesse and Ramond, and Piccinino, as well as the cases previously reviewed by one of us, is not conclusive. In the majority of cases, including the two previously reported by one of us, and the case at present under consideration, the results of bacteriological examination have been negative.

In Landry's original case and in a considerable number of cases reported since, the anatomical changes were reported negative. Many of those cases, however, can not at present be cited as evidence, since they were examined before the present microscopical technique had been introduced. A few cases, however, reported recently demand a brief consideration. Schultz,[13] in an article accessible only in an abstract, speaks of a case of acute ascending paralysis in an alcoholic patient which proved fatal in fifteen days. Nothing was found in the nerves or cord, but no statement is made in the abstract as to the methods employed. Goebel,[14] in a case of seventeen days' duration, found the peripheral nerves normal (osmic acid, Weigert, carmine); in some muscles fresh degeneration of the muscle fibers and occasionally increase of interstitial tissue; in some bundles of the cauda equina focal destruction of nerve fibers in the neighborhood of a vessel distended with blood, with fresh degeneration (Marchi) of some axis cylinders and medullary sheaths; the spinal cord (Weigert, carmine, Marchi), and the anterior roots (Weigert, carmine, osmic acid) were normal. From the decussation of the pyramids to the oculomotor nuclei some slight degeneration in the long tracts and the nerve roots could be detected by Marchi's method. In the discussion Nonne reported a case with marked bulbar symptoms where both the medulla oblongata and the cranial nerves were normal. In both cases Nissl's methods gave no certain anomalies, but Nonne recalled the opinions of Goldscheider and Flatau that we must be cautious in estimating the value of changes found by these methods. It is again uncertain with the incomplete report how far we can regard "no certain anomalies" (keine sicheren Anomalieen) as absolute proof of negative findings. Girandeau and Lévi[15] have recently reported a case of ten days' duration, coming on during convalescence from a typhoid which had been treated with anti-typhoid serum, where after careful use of osmic acid, Pal's method, hematoxylin-eosine, picro-carmine, Nissl's method, neither they nor Nageotte were able to discover any changes.

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[9] Burghart, Charité Annalen, xxii, 1897.
[10] Chantemesse and Ramond, Revue Neurologique, 30 Nov., 1898.
[11] Remlinger, Revue Neurologique ,15 April, 1898.
[12] Rendu, Revue Neurologique, 15 July, 1897.
[13] Schultz, *art. cit.*
[14] Goebel, Neurologisches Centralblatt, 1 April, 1898.

This last case, at any rate, seems to indicate that even with modern technique no anatomical changes were found. To it, and to the others, must be opposed a much greater number of cases where modern methods show definite changes. Accepting these few cases as they stand, they simply show that in rare instances our modern methods are inadequate to reveal the changes produced by the toxic agent which gives rise to acute ascending paralysis. In the great majority of cases, however, our modern methods prove conclusively that there is an acute parenchymatous degeneration of the peripheral motor neurone manifesting itself most markedly at times in the ganglion cell, at other times in the axis cylinder process. Bodin,[16] has claimed that the whole nervous system from the brain to the nerves may be attacked, but this is not confirmed either clinically or pathologically.

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[15] Girandeau and Lévi, Revue Neurologique, 15 Oct., 1898.

[16] Bodin, "Essai sur les paralysies ascendantes aiguës." Thèse de Paris, 1896.

Krewer,[17] however, has advanced a theory, based upon four cases, of which three came to autopsy, which also derives some support from one of the cases already cited reported by Schultz. He found, as we have said, changes in the peripheral nerves indicative of chronic neuritis, together with changes in the cord indicative of acute myelitis. Of his fatal cases three were alcoholic and one also tuberculous, and all three had had influenza shortly before the onset of the acute paralysis. He maintains that Landry's paralysis is the second and third stages of a chronic multiple neuritis, which has extended to the spinal cord *per continuitatem.* In the latter it extends very rapidly, usually upwards, but sometimes downwards, and causes death by involvement of the vital centers in the floor of the fourth ventricle. A new factor in the form of an infectious disease is usually necessary to cause an outbreak of Landry's paralysis in an existing multiple neuritis. Neither in the two cases previously reported nor in the three we bring forward to-day was there any evidence of a pre-existing multiple neuritis. As a rule such a history is absent in the cases reported clinically by other observers, and in many cases with the autopsies the anatomical changes of chronic multiple neuritis have been absent.

Having spoken of the pathological basis of acute ascending paralysis we will now report three cases, one of which came to autopsy, one made a partial recovery, and the third recovered completely.

CASE I. On the fifth of March, 1898, we were asked by Dr. F. W. Anthony, of Haverhill, to whom we are indebted for notes in regard to the patient, to see Mrs. D., who had been ill for about a week.

She was born in Massachusetts, of American parentage, and was between twenty-seven and twenty-eight years of age. Her father had been a dissipated man; her mother had died of valvular disease of the heart, cirrhosis of the liver, ascites, and carcinomatosis. The patient herself had had, in childhood, measles, whooping cough, mumps and scarlet fever. Four years previously, she had an attack of influenza, when she slept for three days and nights, and had to be wakened to take food, immediately falling to sleep again. She had no physician at the time, and was up in about a week. She had always been subject to migraine up to about three years ago, since which time the attacks had been much less frequent. She had one child ten years ago, with normal labor; she had had no miscarriages. Her husband was a very dissipated man, and she had not lived with him for the past five years. She herself had indulged very slightly in alcohol, but never to excess, and it was reported that since leaving her husband she had lived with another man, but no history could be obtained of syphilis, and she had not led a dissipated life. She had had internal strabismus of the right eye since childhood, following a fall into a well. She had always been subject to constipation, and for the past year she had had a peculiar fluttering feeling in the back, but, on the whole, her health had been better for the last six months than it had ever been during her life.

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[17] Krewer, *art. cit.*

On the 26th of February, when she arose in the morning, she noticed that the legs felt stiff and it was hard to get up. She could not go down stairs as well as usual, and after she went out she fell in stepping off the curbstone. On the 27th of February she fell while running across the floor, and subsequently during the day she fell repeatedly while walking, and the legs felt considerably weaker. On the 28th she was able to walk, but she fell more frequently. Dr. Anthony was called in, and he found that the power of abduction was lost in the left leg and the power of adduction was much diminished; abduction and adduction were diminished in the right leg; she was unable to flex the left leg at all, and she had great difficulty in flexing or extending the right leg upon the thigh; the knee-jerks were absent; there was no fever; no impairment of sensibility; no disturbance of the bladder or rectum; vaginal examination was negative.

March 1st. She was wholly unable to walk; the left leg was practically powerless; the right leg was very weak; she could not raise it in bed or abduct or adduct it at all; sensibility was normal; the arms were unaffected.

March 2d. The condition was much the same. There was tenderness on percussion on each side of the lumbar vertebræ.

March 4th. The grasp was diminished in both hands; the right pupil was somewhat dilated; she complained of some disturbance of vision, as if she saw objects through something like a veil; the pupils were normal and reacted to light; she slept poorly and complained of inability to swallow solids and difficulty in swallowing liquids.

March 5th. We both saw her with Dr. Anthony, in consultation. She did not have much pain, but complained of some cramp in the abdomen and of some soreness in the head; the legs felt as if quivering or crawling, and she had some cramp or pain in them. There was a trembling, quivering feeling beneath the skin, in the legs and throat; she slept poorly, thinking constantly but not worrying; she had considerable palpitation and a beating feeling all over. There was no difficulty in talking, but fluids seemed to stick in the throat when she attempted to swallow. The appetite was good; the digestion unimpaired; the bowels and the bladder acted normally; there was no disturbance of the pelvic organs. She had never had any menstrual disturbances. Everything seemed blurred that she looked at, but there was no double vision. She was dressed and sitting in a chair; rather a slight woman, well developed, fairly well nourished and of good color. The right pupil was larger than the left; neither reacted to light or accommodation. Vision in the right eye had always been less than in the left. The visual field was normal; the ophthalmoscopic examination negative. The movements of the eyes, face, tongue and palate were normal. She could make all movements with the arms, but all the movements were weak. The movements of the ribs in respiration were equal, but not up to the normal; the movements of the abdominal muscles were fairly good; she could twitch the legs slightly at the trunk, but she could not flex the thighs upon the body or abduct or adduct the legs; there was a slight movement of rotation outwards; there was no motion at all at the knee-joint, and only a very slight plantar flexion of the feet. Sensation everywhere was good for touch, pain and temperature, but the right leg seemed somewhat hypersensitive to pain. There was considerable tenderness on pressure over the posterior tibial nerves; a slight tenderness on pressure over the nerves in the arm; there was also some tenderness over the vertebræ by the sides of the spine in the mid-dorsal region and over the sacro-iliac synchondrosis. The muscles of the lower leg did not react to a tolerably strong faradic current; the vastus internus reacted to a moderately strong current feebly, less upon the right side than upon the left; the muscles of the arms reacted normally to faradism. The knee-jerk was absent; the plantar reflex was absent on the right and much diminished on the left; the abdominal and epigastric reflexes were normal. Examination of the chest was negative. The splenic dullness extended between three and four centimeters on each side of the costal border. The examination of the abdomen and of the pelvic organs was negative. The temperature was not elevated. A watch was heard at three inches from either ear; the bone conduction was equal; there was a slight weakness of the right levator palpebræ superioris.

March 6th. The grasp of both hands was much diminished, especially on the left side. She swallowed liquids with the greatest difficulty. She complained of headache, most severe in the occipital region. The abdominal muscles were not markedly affected. Sensation was apparently unimpaired. The right pupil was dilated and the vision was less distinct with the right eye. She was unable to move either leg. She was taken to the hospital.

March 7th. The tongue was protruded to the left. She had to be catheterized last night and this morning; she could take a small amount of nourishment by mouth. The feet and legs pained her a good deal during the night. She was given nutrient enemata every three hours.

March 8th. Some difficulty in articulation. Incontinence of urine twice during the night, but she passed urine naturally afterwards; later she had to be catheterized. She felt drowsy much of the time; the feet and legs pained her considerably during the night; the pupils were nearly equal, responding sluggishly to light. There was some tenderness on each side of the cheek, over the rami of the jaws and at the sides of the head in the parietal region.

March 9th. Vision remained about the same. The arms were in about the same condition. She felt drowsy; had a good deal of abdominal distress during the night; had a prickly sensation in the feet and arms during the night; she had a choking spell which exhausted her considerably.

March 10th. No incontinence during the night. She had great pain in the abdomen, relieved by $\frac{1}{8}$ of a grain of morphine. She was considerably weaker and drowsy. She had some trouble with mucus in the throat, which she coughed up at intervals. She had pricking, tingling sensations in the hands and arms, and increasing difficulty in articulation. She grew considerably weaker, and had choking spells; the pupils were about equal. The enema was not retained this afternoon.

March 11th. Nutrient enemata were retained with difficulty, some not at all. Amount of urine in twenty-four hours, thirty-five ounces.

March 12th. Died.

Autopsy March 14th, 1898.

Body of a woman about 28 years of age, well developed, and nourished. Rigor mortis present.

Spinal cavity alone opened.

Cord on examination showed nothing abnormal. Hardening in formalin.

Microscopical examination: Sections from the lumbar enlargement show a marked dilatation of the blood vessels of the meninges, but without hemorrhagic extravasation. The nerve roots, in sections stained by Marchi's method, show diffuse degeneration of fibers, which is fairly extensive, and involves perhaps one-third of the fibers. This degeneration is somewhat more marked in the anterior than in the posterior nerve roots. The blood vessels in the nerve roots show the same marked dilatation and congestion which is present in the meninges with some hemorrhage among the nerve fibers. The blood vessels of the cord itself are also greatly dilated, particularly in the region of the anterior horns. A few of the vessels retain their normal size. There are no hemorrhages into the substance of the cord, nor is there any purulent infiltration. Sections stained by Marchi's method show a diffuse degeneration all through the white substance of the cord, and in the commissural fibers of the gray matter. Many of the nerve sheaths show the presence of fat, and sometimes they are en-

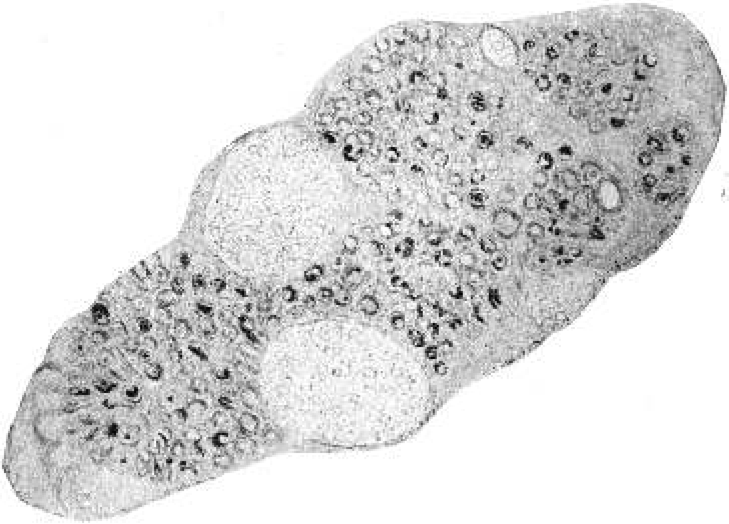
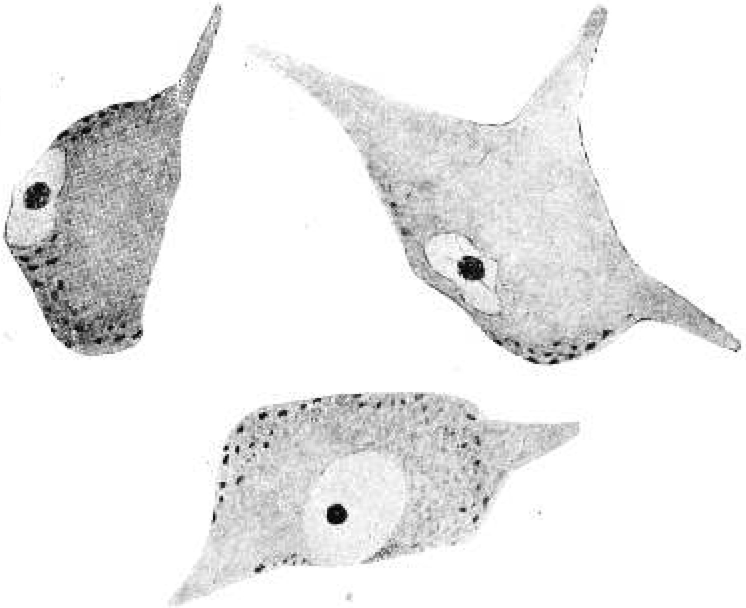


Fig. I. Low power. Zeiss, AA obj. No. 4 ocular. Nerve root, lumbar region, Marchi's method. Shows enormously dilated blood vessels, and degenerated nerve fibers.

tirely replaced by fat. Occasionally the axis cylinder is replaced by fat, though this occurs less frequently than the degeneration of the myelin sheath. Rarely, both the axis cylinder and the sheath have undergone degeneration. The large nerve cells of the anterior horns are not decreased in number. They show no distinct fat globules, but in many of them there is a



Figs. II, III and IV. High power. Zeiss, oil immersion 1-16 obj. No. 4 ocular. Nerve cells from anterior horn of lumbar region. Nissl's stain, showing dislocation of nucleus, disappearance of nuclear membrane and destruction of protoplasmic granules of cell bodies. The nucleus in one of the figures appears to project beyond the cell wall.

single large group of pigment granules. The amount of pigment in these cells is greater than is normal at the age of the patient. In sections stained by methylene blue and eosin, and by Nissl's stain, few of the nerve cells appear normal. In most sections two or three cells with distinct granules are all that can be found, the others showing marked changes in the granules and nuclei. In many of the large cells which are cut through the processes, no nucleus or nucleolus can be found, though sometimes there is to be seen in the cell body a clearer spot, which evidently represents the location of the nucleus.

In other cells the nucleus stains faintly and homogeneously by Nissl's method, the nuclear membrane is broken or absent, and the nucleolus is faintly stained. In other cases the nuclear membrane is indistinct, the nucleus clear, however, while the nucleolus is larger than normal, stains a purplish color, and is vacuolated. In almost all the cells where the nucleus is seen it lies not in the center of the cell body, but is dislocated towards the periphery. Occasionally it lies so close to the cell wall that no protoplasm can be seen between it and the cell wall, and at times it even forms a projection in the cell wall, as though it were being extruded. In most of the cells spoken of above in which no nucleus can be seen, the clear area within the cell body, which represents the nucleus, lies excentrically. The protoplasmic granules of the cell body, even when present, do not stain sharply, but are ragged in outline, and vary a good deal in size. Occasionally a cell is found which has practically normal Nissl granules. In most of the cells, however, these granules cannot be made out at all, but the cell protoplasm stains homogeneously, or is filled with fine granular dust, with a few granules at the periphery of the cell. In the sections stained by Nissl's method, many of the cells show clear, yellowish areas in the cell body, caused by the presence of pigment granules. The dendritic processes of the cells are not broken, but can be traced as far as is usual. The granules in these processes, however, are fewer than normal; but occasionally they appear distinct, and of normal size.

The cells of the posterior horns of gray matter show a few granules at the periphery of the cell, and the nucleus and nucleolus are well preserved; the cells in general showing very little change.

In the dorsal region of the cord the same general changes are seen, particularly the engorgement of the blood vessels. By Marchi's method the same diffuse degeneration in the white matter is found. The cells of the anterior horns are as a rule more normal in appearance than those in the lumbar region. The nucleus and nucleolus show only rarely any marked dislocation. In some of the cells the nucleus has taken a faint blue tinge. The nucleoli are irregular in outline. The protoplasmic granules in many of the cells are normal, while in other cases they are fewer in number than normal, being seen chiefly at the periphery of the cell, while the cell body is stained faintly and homogeneously. The cells of the column of Clarke have fewer protoplasmic granules than normal, and these are chiefly at the periphery of the cell, but the changes are not marked.

In the cervical region there are found the same lesions in the white substance of the cord, and in its membranes, that were seen lower down. There are also degeneration of nerve cells, which are of the same character as those seen in the lumbar region, and fully as intense, but a smaller proportion of nerve cells are affected.

Sections from various levels, stained by Gram's method for organisms, failed to show their presence.

A cross section of the sciatic nerve, treated by Marchi's method, shows extensive fatty degeneration of the myelin sheaths of a good many of the fibers. The nerve fibers appear somewhat swollen, and where no fat is seen, they are exceedingly granular, and the axis cylinders cannot be made out. In the longitudinal sections of the same nerve the osmic acid has penetrated poorly, but in the places where it has penetrated the myelin sheaths show an irregular, but extensive degeneration, which has affected most of the individual fibers.

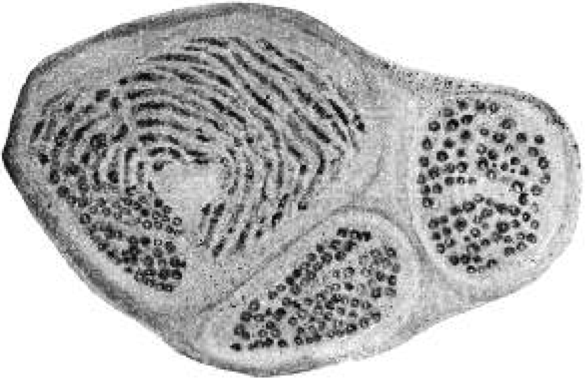


Fig. V. Low power. Zeiss, AA obj. No. 4 ocular. Portion of cross section of sciatic nerve. Marchi's method. Degenerated nerve fibers.

Anatomical Diagnosis:

Parenchymatous degeneration of nerve cells of anterior horns, with destruction of protoplasmic granules, and nuclear changes.

Parenchymatous degeneration of anterior and posterior nerve roots, and of peripheral nerves.

Dilatation of blood vessels of anterior horns of the cord, of the meninges, and of the nerve roots.

To recapitulate, a fairly healthy young woman was suddenly attacked with weakness and later complete paralysis of the legs, extending to the arms, eyes and throat, with some involvement of the sphincters, slight disturbances of sensibility (pain, tenderness and paresthesia), loss of reflexes, and diminished reaction to faradism; the trouble resulting fatally in fifteen days. The autopsy showed degeneration of the nerve cells in the anterior horns, degeneration of the anterior and posterior nerve roots, and degeneration of the peripheral nerves, with dilatation of the blood vessels in and about the cord.

CASE II. For permission to report the second case, we are indebted to the kindness of Dr. Henry Jackson, in whose service the patient was at the City Hospital.

A. M., twenty-four, single, a laborer, a native of Cape Breton. His father died of rheumatism, his sister of phthisis. The patient had always been in good health and had rarely indulged in alcohol.

On the 18th of September, 1896, he was taken with severe headache, nausea and vomiting. The next morning he felt numb and weak and noticed a partial loss of power in both legs below the knees; he was able to go about the room with difficulty. The loss of power and numbness steadily increased and the next day he was unable to get out of bed, and felt feverish. There was no pain except when he was raised to a sitting posture, when there was some pain in the back. The appetite was poor; the bowels acted normally, but on the 21st, when he took a dose of salts, and on the 22d, when he was admitted to the hospital, he had an involuntary movement. There was no retention of urine. He was well developed and nourished, with an apathetic expression. The conjunctivæ were slightly injected; the pupils were equal and reacted normally; the pulse was regular and of good strength and volume; the tongue was protruded straight. Examination of the chest and abdomen was negative, except that the abdominal muscles were found to be weak; the abdominal reflexes were present; there was no tenderness of the back. The arms were normal. There was marked weakness of both legs; he was able to move the knees and feet only very slightly. The sensibility to pain was considerably diminished; the sensibility to touch and muscular sense was normal. The cremasteric reflexes and the knee-jerks were absent, the plantar reflex diminished; the muscles of the legs were flabby; the urine was negative. The temperature was 103° F.

On the 23d there was retention of urine with considerable pain so that he had to be catheterized. The weakness increased so that he could not turn in bed, and that day he had an involuntary movement of the bowels. The temperature was lower.

On the 24th he was more apathetic; the left side of the mouth drooped; he felt weaker; there was no pain, and sensibility to touch was normal.

On the 25th the temperature had fallen to normal, where it remained. The plantar reflex was absent; the arms were very weak; the left mouth drooped still more; he vomited and had many loose and involuntary stools. A lumbar puncture was made; one drop of clear fluid and a little blood were obtained, but no pathogenic organisms were found. He still had to be catheterized.

On the 29th he seemed brighter. An ophthalmoscopic examination was negative. He was able to pass urine voluntarily, but the stools were still loose and involuntary.

On the 3d of October there was so much pain in the legs as to prevent sleep. The appetite was good, but he was costive.

On the 9th, sensibility was still normal; there was no nerve tenderness. The paralyzed muscles were soft and flabby; the abdominal reflex was lost.

On the 13th he began to gain strength in the arms and gradually recovered control of the bowels, although very costive; he also had much rectal pain; there was complete loss of knee-jerk, complete loss of power and marked atrophy of the legs, and he could turn in bed only by the help of his arms. He required a daily enema. This condition remained until the 10th of November, when his friends took him to his home in the Provinces. Nothing has been heard from him since.

To recapitulate, a healthy man is suddenly seized with weakness of the legs, increasing to complete paralysis and extending to the abdominal muscles, the arms and the left side of the face, with loss of reflexes, loss of control of the sphincters, and hypoalgesia with no hypoesthesia. Later on there was marked muscular atrophy, and recovery was incomplete.

CASE III. The following case was seen in the service of Dr. C. F. Folsom, at the City Hospital, to whom we are indebted for permission to report the case.

William I., a colored laborer, single, aged twenty, a native of Massachusetts, was admitted to the hospital the 2d of November, 1898. The family history was negative. The previous history was good, except for some muscular rheumatism in the right leg. He was a man of good habits. On the 17th of October he was in excellent health, except for some slight pain in the lumbar region, which he had had for two years. On the 19th, he woke with a heavy headache, general malaise and loss of appetite. The pain in the lumbar region was worse and he could not lie in any position that would relieve it. There was no pain or tenderness in the legs or excessive sweating. The legs began to feel numb, the sensation beginning at the tips of the toes and progressing upward daily. He began to lose power in both legs at the same time, the trouble rapidly increasing; the muscles of the trunk soon became affected; the respiratory muscles followed, so that he could not take a deep breath, and when he tried to do so the air seemed suddenly forced out; the arms also had grown steadily weaker. Just before admission to the hospital, he had some pain in the sternomastoid muscles. On the morning of admission, he found he could not swallow toast. There was no trouble in articulation, and no fever. He had normal control of the sphincters, but the bowels had not moved for three days. On admission, he was found to be well-developed and nourished; the mental faculties clear; the speech not affected; the tongue was protruded straight; the pupils were equal and reacted normally; the pulse was rapid, regular, of good volume and tension. Examination of the chest and abdomen revealed no abnormalities in regard to the internal organs; the knee-jerks were absent; the cremaster and plantar reflexes diminished; the sensibility to touch and temperature was everywhere normal; there was no hyperesthesia or tenderness over the nerves. He could flex the legs upon the thigh and the thigh upon the abdomen very feebly and with great effort. He could not raise the body while lying in bed. The hand-grasps were very weak. He could turn with much difficulty to one side—better to the right than to the left—and he could move the head without difficulty. The voluntary movements of the facial muscles were normal; no noticeable atrophy could be perceived. The extremities were cold. On the 6th, the weakness had increased, but he had had no pain. For the first time he noticed great difficulty in swallowing water, all that he took being regurgitated. The speech was thicker, slower and more inarticulate, and it was a great effort to speak. There was no special difficulty with the respiration. The masseters, even when he tried to close the jaws tightly, felt soft and flabby. There was double facial paralysis and anesthesia of the pharynx. The pupils were equal, but reacted sluggishly; he thought, however, that he could move the toes somewhat better. The weakness steadily increased until, on the 10th, it had become almost impossible for him to swallow any food and it was decided to resort to tube feeding. There was not much difficulty, however, in respiration; the sensibility was unimpaired. The electrical reactions were unfortunately not tested. On the 10th he began to improve, and on the 11th, he was able to swallow a little food. The speech was less thick, but he was unable to close the eyelids or to pucker up the lips. There was no loss of control of the sphincters. He had some pain in the muscles after lying in one position, but he was able to move the toes slightly. The temperature during this period was, for the most part, normal, although occasionally, in the afternoon, there was an elevation of one or two degrees.

On the 15th the gain had become marked; he could move the legs in bed, especially in extension; he had no trouble in swallowing; the grasp of the hands was stronger; he could close the eyelids and also pucker up his lips to whistle; the voice was not much affected, and the masseters felt firmer. From that time he gained steadily.

On the 23d a blood-count showed 7,600 white corpuscles.

On the 27th of November he could whistle, close his eyes tightly, and he sat up in a chair by the bed. He had some pain in the left calf.

On the 3d of December he could sit up straight in bed and get from the chair into bed without help.

On the 9th of December, the calves were still flabby, but the arms were of good strength.

By the 16th he was able to walk fairly well, although the legs had not regained their full size, and on the 22d he was able to leave the hospital. He has since made a complete recovery.

To recapitulate, a healthy young man is suddenly attacked with weakness of the legs, increasing to complete paralysis and extending to the abdominal and respiratory muscles, the muscles of the face and the muscles of mastication and deglutition. The reflexes were lost. The sensibility was not markedly impaired. There was later some muscular atrophy with complete recovery.

These three cases present three different terminations of Landry's paralysis and also raise certain questions as to the clinical features of the disease. The pathological lesions show the close connection of the affection with acute multiple neuritis and with acute poliomyelitis. Pathologically, it is both;

clinically, however, it differs somewhat in its manifestations from the ordinary type of either disease. The course of ordinary anterior poliomyelitis is quite different; beginning as Landry's paralysis may, with signs of acute febrile disturbance, it affects a considerable number of muscles at once, and is not progressive in character; the abdominal and respiratory muscles and those supplied by the cranial nerves are seldom affected and the sphincters remain intact. Sensory disturbances, except some painful sensations, are lacking. In neuritis, on the other hand, sensory disturbances are more marked. The trouble is less often progressive, and the cranial nerves are usually spared. In neither affection do we note the steady progress as in Landry's paralysis, from the feet upward until the cranial nerves are involved. Whether Landry's paralysis may extend downwards, as it is said to have done with Cuvier, requires further evidence.

Although the poison which produces Landry's paralysis is, like lead, selective, affecting the motor neurone, we must remember that sensory disturbances, amounting to anesthesia of the feet and finger-tips, were present in Landry's original case. The sensory disturbances are much less marked, but they are by no means uncommon, and the presence of degeneration in the posterior nerve roots in the case presented today gives a partial explanation. The neuritis which is so often present may affect sensory fibers as well as motor, although the affection of the motor fibers predominates.

The disturbance of the sphincters, noted in two of the cases, is not usual, but it may be explained in part by the weakness of the abdominal muscles preventing effective straining. It has been met with, although rarely to a marked degree, in cases otherwise presenting the symptoms of Landry's paralysis so that its presence can hardly be regarded as disproving the diagnosis.

In the two cases that recovered, there was considerable muscular atrophy, and in the fatal case there was a diminished reaction of the muscles to faradism. One of the striking features of Landry's paralysis, as given by Landry himself, and as restated or confirmed by the majority of writers since, is the existence of complete paralysis without muscular atrophy and without changes in the electrical reactions. Westphal[18] in 1876, defining Landry's paralysis, stated that it was characterized by its progressive ascending course with fatal termination, by the fact that the paralyzed muscles retained their electrical excitability intact, and by the negative results of autopsy. The progressive ascending course is essential, but, as these two cases show, the termination is not inevitably fatal and the results of autopsy are not negative. The question of atrophy and electrical changes is still open.

With the establishment of the anatomical changes in Landry's paralysis as a degeneration of the peripheral motor neurone, we must admit *a priori*, if our modern notions as to the symptomatology of nervous affections be correct, that muscular atrophy and altered electrical reactions will inevitably result. Muscular atrophy and marked electrical changes, however, require a certain time for development, and in Landry's paralysis the patient may be dead before they ever become marked. The ordinary methods of clinical examination, moreover, do not favor the detection of slight changes in electrical reactions, especially quantitative estimates. In a considerable number of cases where the paralysis was acute, ascending and progressive, especially in cases that did not end fatally, muscular atrophy and electrical changes have been noted. In Burghart's case already cited,[19] which recovered from Landry's paralysis to die of tuberculosis, reaction of degeneration was observed. It is easier to believe that slight electrical changes have been overlooked than that a pronounced degeneration of the peripheral motor neurones can exist without some changes. In severe cases that recover, therefore, atrophy and electrical changes are to be looked for, but many cases die before atrophy becomes marked.

DISCUSSION.

Dr. F. W. Langdon said that, with reference to the sensory symptoms in Landry's paralysis, he had had the opportunity of seeing a case within the past year in which the diagnosis was confirmed by the fatal outcome. It was in the person of a man weighing over 200 pounds, who was connected intimately with the brewing business and drank freely of beer. He had for three or four days before Dr. Langdon saw him numbness in his fingers and hands. He had no symptoms in the legs, and rode about, standing on the platforms of cars because he smoked. When Dr. Langdon saw him he had this distinct numbness in the arms, but could feel the ordinary touch of the hand. There was a very suspicious weakness of respiration, and the development of Landry's paralysis was feared. There was also tenderness on pressure over the principal nerve trunks, but no spontaneous pain. A guarded prognosis was given and the family was frankly informed that the outcome could not be foretold, and that if the disease ascended any higher it would probably be fatal. The man died two days later. The probabilities are that the symptoms of numbness, etc., are so slight as compared with other symptoms that they are often overlooked.

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[18] Westphal, Archiv für Psychiatrie, vi., 765, 1876.

[19] Burghart, *art. cit.*

Dr. C. E. Riggs stated that he had under his care a case of multiple neuritis that was rather anomalous in its development. For the first three or four days the patient complained of weakness in the lower extremities, extending later to the upper extremities, and he was brought into Dr. Riggs' office absolutely unable to make any voluntary motion. Perhaps a week afterward he commenced to suffer severe pain, and the evolution of the case made it clear that it was a form of multiple neuritis. Another point that quite impressed Dr. Riggs was that at one of his visits he observed that the patient had a very rapid pulse, with perfect regularity of the respiration and heart beat. In the evening these symptoms increased and a condition of nervous shock developed, so that Dr. Riggs thought that the man would not live through the night. He was placed under opiates and began to rally, and is now getting along very nicely.

II. ALBUMINURIE POST-PAROXYSTIQUE DANS L' EPILEPSIE CONVULSIVE (Post-Paroxysmal Albuminuria in Convulsive Epilepsy). M. Lannois and L. Mayet (Lyon Médical, 31, 1899, p. 365).

In a study of fifty patients involving about four hundred urine analyses, the authors found albumin in the urine after fifty-five per cent. of epileptic paroxysms and suggest that its presence may be of signal value in excluding simulated epilepsy and hysterical convulsions. After mention of several explanations of the presence of albumin, they conclude that it is due to circulatory disturbance induced by the fit and note with Voisin and Péron that it is the patients who become cyanotic in the attack who afterward void albuminous urine. This post-epileptic albuminuria is transitory and in direct relation to the intensity of the asphyctic stage of the paroxsym. PATRICK.